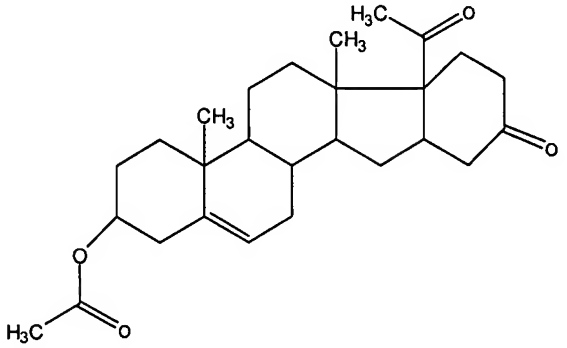
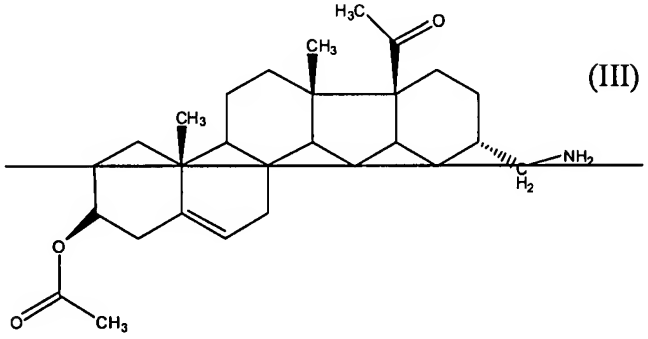
Amendments to the Specification:

Please replace paragraph 42 of the published specification with the following amended paragraph:

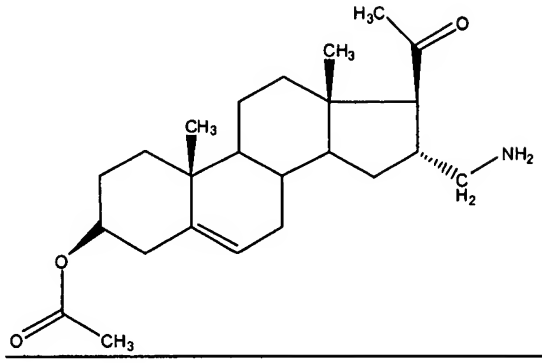
[0042] wherein the X and R_1-R_6 groups are as defined above. In a preferred embodiment, the compound is selected from the group consisting of:



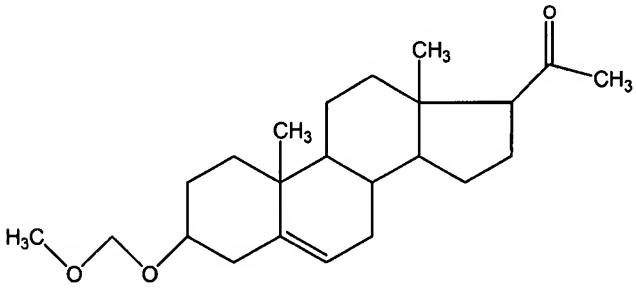
(II)



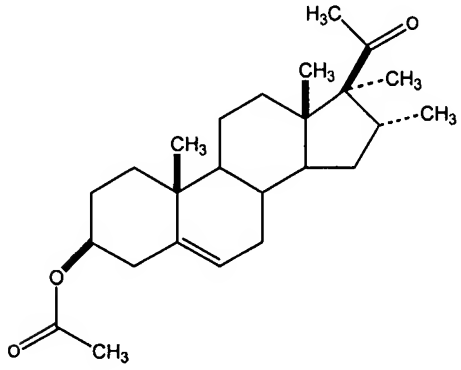
(III)


H3C
O
CH3
CH3
NH2
C
H2
O
O
CH3

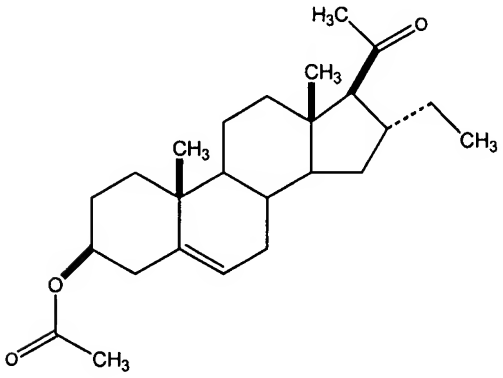
(III)


O
CH3
CH3
CH3
H3C
O
O

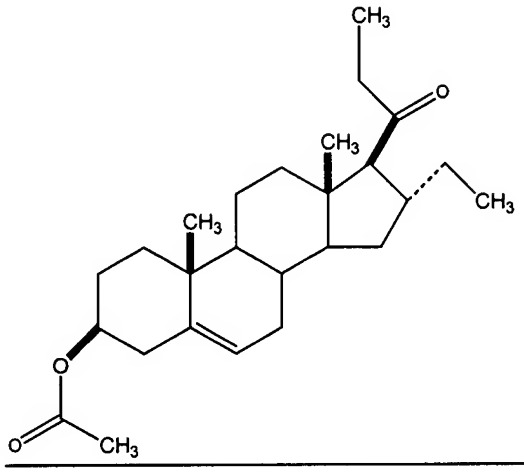
(IV)


H3C
O
CH3
CH3
CH3
CH3
O
O
CH3

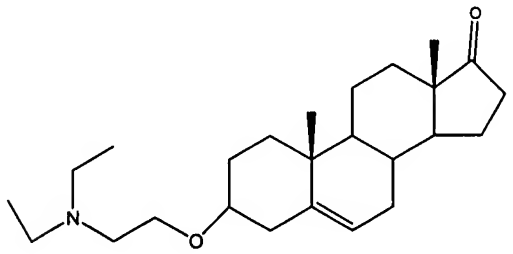
(V)


H3C
O
CH3
CH3
CH3
O
O
CH3

(VI)



(VII)



(VIII)

Please replace paragraph 79 of the published specification with the following amended paragraph:

[0079] Also disclosed by the present invention is the fact that wild-type melanogenic cells with normal P protein function secrete some tyrosinase, and that compounds that increase secretion of tyrosinase in a P protein dependent manner also inhibit melanogenesis. Accordingly, the present invention provides novel methods of screening for compounds that increase melanogenesis by increasing the function of P protein. For purposes of this application, compounds that increase the function of P protein and compounds that decrease the function of P protein are collectively referred to herein as "compounds that affect the function of P protein." Still another aspect of the invention is a method of screening for compounds that increase melanogenesis by mimicking the function of P protein. For purposes of the invention, [[?]] "compounds that mimic the function of P protein" [[?]] are compounds that are not P proteins, yet when they are administered to, or incubated with, melanogenic cells that do not contain P protein, they serve to restore at least in part the correct targeting of tyrosinase to the melanogenic membrane. Melanogenic cells that do not contain P protein may be cells that do not express P protein transcripts (such as melan-p

cells, described herein) or melanogenic cells that do not express a functional P protein gene product.

Please replace paragraph 88 of the published specification with the following amended paragraph:

[0088] Because growing P-protein-deficient melanocytes in the presence of high levels of tyrosine ~~tyrosinase~~ in the medium can partially rescue the P-protein-deficiency, it is preferable, but not necessary, that a screen for inhibitors of melanogenesis is carried out in the presence of low amounts of tyrosine in the media, e.g., 0.01-0.05 mM [[μm]] tyrosine, more preferably 0.014-0.03 mM [[μm]] tyrosine.

Please replace paragraph 112 of the published specification with the following amended paragraph:

[0112] Another effect of inhibited melanogenesis caused by a mutation which inhibits P protein function is the disruption of a high molecular weight complex comprising tyrosinase, TRP-1 protein, and TRP-2 protein (Orlow, S. J. et al., 1994, supra). For purposes of the present invention, the term "high molecular weight complex" is defined as a group of proteins bound to each other via covalent and/or non-covalent bonds that remain associated with each other during non-denaturing gel filtration, HPLC, or sucrose gradient sedimentation and have an apparent molecular weight of between about 200 kD and about 700 kD. In wild-type melanogenic cells, this "melanogenic complex,"[[?]] which is associated with the melanosome, contains a significant fraction of the cells' complement of tyrosinase, TRP-1 protein and TRP-2 protein. In melanogenic cells inhibited for melanogenesis by inhibition of P protein function, very little of any of these proteins is found in high molecular weight complexes.

Please replace paragraph 196 of the published specification with the following amended paragraph:

[0196] Non-limiting examples of compounds that cause an alteration in late endosomal/lysosomal trafficking of cholesterol include U18666A ~~(formula VII)~~ (formula VIII) and its derivatives ~~(e.g., formulae II-VI)~~ (e.g., formulae II-VII), either alone or in combination. Useful examples of U18666A derivatives include CP-598755-01 (formula III), CP-602367

(formula IV), CP-352369 (formula II), UK-204039 (formula V), UK-204041 (formula VI), and UK-204042 (formula VII). Generic formula I presented herein is derived from U18666A and its derivatives presented herein (also see FIG. 16).

Please replace paragraph 220 of the published specification with the following amended paragraph:

[0220] The term "late endosomal/lysosomal trafficking" is used herein to refer to the movement of proteins, lipids, or other compounds between different cellular compartments. These locations include the movement of such compounds from the late endosome to the lysosome, from the lysosome to the late endosome, from the late endosome or lysosome to the trans Golgi network, and from the trans Golgi network to the late endosome or ~~lsysome~~ lysosome.

Please replace paragraph 340 of the published specification with the following amended paragraph:

[0340] The results are presented in FIG. 19 (small granule fractionation) and ~~FIG. 8~~ FIG. 20 (large granule fractionation). These data indicate that the localization of tyrosinase is markedly affected by exposure to U18666A. For example, in FIG. 19 a shift in the distribution of tyrosinase is evident in comparing fraction 5 of the control and U18666A samples. The change in the distribution of tyrosinase is even more evident in the large granule fractionation study (see FIG. 20, fractions 6-8, for the large granule fraction control (LGF-C) and large granule fraction U18666A (LGF-U18)).